Fifty Men, 3510 Marathons, Cardiac Risk Factors, and Coronary Artery Calcium Scores

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ABSTRACT

ROBERTS, W. O., R. S. SCHWARTZ, R. F. GARBERICH, S. CARLSON, T. KNICKELBINE, J. G. SCHWARTZ, G. PEICHEL, J. R. LESSER, K. WICKSTROM, and K. M. HARRIS. Fifty Men, 3510 Marathons, Cardiac Risk Factors, and Coronary Artery Calcium Scores. *Med. Sci. Sports Exerc.*, Vol. 49, No. 12, pp. 2369–2373, 2017. **Introduction/Purpose**: Many male marathon runners have elevated coronary artery calcium (CAC) scores despite high physical activity. We examined the association between CAC scores, cardiovascular risk factors, and lifestyle habits in long-term marathoners. **Methods**: We recruited men who had run one or more marathons annually for 25 consecutive years. CAC was assessed using coronary computed tomography angiography. Atherosclerotic cardiovascular disease risk factors were measured with a 12-lead ECG, serum lipid panel, height, weight, resting blood pressure and heart rate, and a risk factor questionnaire. **Results**: Fifty males, mean age 59 ± 0.9 yr with a combined total of 3510 marathons (median = 58.5, range = 27–171), had a mean BMI of 22.44 ± 0.4 $kg \cdot m^{-2}$, HDL and LDL cholesterols of 58 ± 1.6 and 112 ± 3.7 $mg \cdot dL^{-1}$, and CAC scores from 0 to 3153. CAC scores varied from 0 in 16 runners to 1–100 in 12, 101–400 in 12, and >400 in 10. There was no statistical difference in the number of marathons run between the four groups. Compared with marathoners with no CAC, marathoners with moderate and extensive CAC were older ($P = 0.002$), started running at an older age ($P = 0.003$), were older when they ran their first marathon ($P = 0.006$), and had more CAD risk factors ($P = 0.005$), and marathoners with more CAC had higher rates of previous tobacco use ($P = 0.002$) and prevalence of hyperlipidemia ($P = 0.01$). **Conclusion**: Among experienced males who have run marathons for 26–34 yr and completed between 27 and 171 marathons, CAC score is related to CAD risk factors and not the number of marathons run or years of running. This suggests that among long-term marathoners, more endurance exercise is not associated with an increased risk of CAC.
Key Words: ENDURANCE EXERCISE, LONG-TERM RUNNING, HEART DISEASE, CORONARY ARTERY DISEASE

Marathon running and the effects of long-term endurance activity on the heart are topics of past and present concern. In 1976, Bassler hypothesized that anyone able to complete a 42-km running race, the standard marathon distance, would not die of CAD (1,2). The "Bassler hypothesis" was short lived, when Noakes presented six cardiac deaths during marathon participation at a New York Academy of Sciences meeting on the marathon in 1976 (8). Despite this evidence, the notion that marathon running was protective against CAD persisted in the lay literature for many years.

Although marathon running is generally considered good for cardiovascular health and fitness, little is known about coronary artery plaque formation in consistent long-term male marathon participants. There are reports of increased coronary artery calcium (CAC) in male marathon runners, raising the possibility that extreme amounts of exercise increase coronary atherosclerosis. Möhlenkamp et al. (6) found CAC scores (CACS) greater than expected based on CAD risk factors in men who had run at least five marathons in the previous 3 yr and significantly higher scores than matched controls ($P = 0.02$), possibly because their risk factors had recently been improved after adopting an active lifestyle. We previously reported more coronary plaque volume measured by coronary computed tomography angiography

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Submitted for publication January 2017.
Accepted for publication July 2017.

0195-9131/17/4912-2369/0
MEDICINE & SCIENCE IN SPORTS & EXERCISE®

DOI: 10.1249/MSS.0000000000001373

(CCTA) in men running one or more marathons a year for 25 consecutive years than in a sedentary, age-matched comparison group (12). However, this same study did not find a statistically significant difference in the number of CAC lesions in the marathoners compared with the sedentary controls (12). By contrast, our study of 26 long-term women marathoners compared with sedentary women showed minimal CACSs, lower coronary artery plaque prevalence, and less calcified plaque volume (11). In the five women in this study with coronary artery plaque, developing lesions appeared to be related to older age and more cardiac risk factors rather than volume of running (11).

The present study reexamines our previous report of increased CAC volume in long-term male marathon runners to determine the relationship between their CACS and their CAD risk factors and lifestyle choices.

METHODS

Male runners who had participated in 25 consecutive Twin Cities Marathon races (St. Paul, MN) were recruited and provided informed written consent as approved by the Institutional Review Board of Abbott Northwestern Hospital (Minneapolis, MN).

CCTA was performed using a Siemens Dual Source or FLASH CT (Siemens, Malvern, PA) with a minimum x-ray dose protocol per standard clinical best practice. At or within 14 d of the CCTA, a 12-lead ECG; serum total, LDL, and HDL cholesterol; triglycerides; and creatinine were determined in a clinical laboratory using standard techniques. Resting blood pressure and heart rate, height, and weight were measured. Subjects completed a lifestyle questionnaire to determine cardiac risk factors and habitual physical activity. CAD risk factors were defined as male sex with age >45 yr, any family history of CAD, past or present tobacco use, history of hypertension, history of hyperlipidemia, history of diabetes mellitus, and body mass index (BMI) >25 $kg \cdot m^{-2}$ (overweight or obese weight classification). Subjects were not included if they reported an allergy to x-ray contrast or had a serum creatinine ≥2.0. Scans were scheduled at least 2 wk before or after a marathon race to avoid using a potentially nephrotoxic agent in a hypohydrated subject.

A validated, commercial software system (Vitrea, Vital Images, Minnetonka, MN) was used to characterize calcium scores by Agatston scoring method and visually estimated percent stenosis. The amount of CAD was determined based on CAC with 0 lesions, no plaque; 1–10 lesions, minimal; 11–100 lesions, mild; 101–400 lesions, moderate; and >400 lesions, extensive. For the risk stratification analysis of the cohort, the minimal and mild groups were combined and labeled low CAC. Coronary artery stenosis was graded qualitatively as absent, minimal (1%–25%), mild (25%–49%), moderate (50%–69%), and severe (≥70%).

Descriptive statistics for each variable were calculated (mean and SE values for continuous variables and counts and proportions for categorical variables). Variables with skewed distributions were log-transformed, and statistical tests were run using both the untransformed and transformed variables. If there were no changes in the conclusions, the untransformed variables and corresponding *P* values were reported to simplify the interpretation. ANOVA was used to test for differences in the continuous variables of interest between the four CACS groups, and chi-square tests were used to test for differences in the categorical variables between the four CAC groups. CAC was split into categories because the distribution was highly right skewed and was not improved by log-transforming. In addition, two-sample *t*-tests/chi-square tests were used to test for differences in the variables of interest between marathoners with no CAC and marathoners with any CAC, and Spearman's correlation was used to look at the overall trend between continuous variables of interest and CAC. *P* values <0.05 were considered statistically significant, and *P* values in ANOVA pairwise comparisons were adjusted using the Tukey–Kramer method. SAS version 9.4 (SAS Institute, Cary, NC) was used for analyses.

RESULTS

The marathon runners' ages, running characteristics, health measures, and risk factors for the entire cohort and for the subcohorts stratified by CAC ranges and the overall correlations are shown in Table 1. The 50 male runners recruited from the pool of 60 men who met the inclusion criteria were on average 59 yr old, ranging in age from 46 to 77 yr. These men had completed a total of 3510 marathons with a median of 58.5 each (range 27 to 171, mean = 70). On average, they started running at age 28 yr, ran their first marathon at 31, and had been running for 26 to 55 yr. Although the marathoners were lean as a group, with a mean BMI of 24.44 $kg \cdot m^{-2}$, their BMI ranged from 20 to 33. Their average HDL and LDL cholesterol levels were 58 and 112 $mg \cdot dL^{-1}$, respectively. There were four runners with HDL cholesterol <40 $mg \cdot dL^{-1}$, two runners from the no CAC group, and two runners from low CAC group. Ten of the runners were being treated with statin medications, and 24 were current (2) or past (22) tobacco smokers. Marathoners with extensive CAC reported more pack-years of smoking ($P = 0.009$). None had diabetes mellitus. The ECG showed normal sinus rhythm or sinus bradycardia, and many runners had changes consistent with "athlete's heart"; there were no ECG showing ischemic cardiac damage. When assessing the correlation between running characteristics and CAC score, the age starting running and age of first marathon were positively correlated with CAC. This means that as the age started running and age of the first marathon increased, CAC generally increased as well. With regard to risk factors, age, pack-years of smoking, LDL cholesterol, and total number of risk factors were also positively associated with CAC score.

As a whole, this cohort was physically active on a year around basis. Of the 50 men in the cohort, 43 ran 12 months

TABLE 1. Differences in age, running characteristics, and health measures by CACS.

Mean ± SE (range)	All Participants	Zero CAC (0)	Low CAC (1–100)	Moderate CAC (101–400)	Extensive CAC (>400)	P^a	Spearman's Correlation ρ (P)
N	50	16	12	12	10		
CAC score	273.97 ± 77.39 (0–3153)	–	36.16 ± 9.27	239.52 ± 30.99	1039.06 ± 274.64	**0.0047***	
Age (yr)	59.44 ± 0.94 (46–77)	55.38 ± 1.38[b]	58.17 ± 1.46[b,c]	62.17 ± 1.86[c]	64.20 ± 2.06[c]	**0.0018***	**0.51 (0.0002)**
Age started running (yr)	28.08 ± 1.19 (7–48)	22.81 ± 1.98[b]	27.17 ± 1.84[b,c]	31.17 ± 2.43[c]	33.90 ± 2.09[c]	**0.0028***	**0.50 (0.0002)**
Age of first marathon (yr)	31.74 ± 0.96 (19–48)	28.00 ± 1.55[b]	30.17 ± 1.59[b,c]	34.83 ± 1.92[c]	35.90 ± 1.93[c]	**0.0055***	**0.47 (0.0005)**
Total marathons completed[e]	70.20 ± 5.57 (27–171)	78.63 ± 11.46	56.75 ± 5.70	79.00 ± 12.58	62.30 ± 12.71	0.3813	−0.10 (0.5034)
BMI (kg·m^{-2})	24.16 ± 0.41 (20–33)	23.27 ± 0.58	24.27 ± 0.96	24.91 ± 0.65	24.56 ± 1.19	0.4772	0.22 (0.1167)
Pack-years of smoking, includes never-smokers	5.86 ± 1.23 (0–36)	1.94 ± 1.06[b]	2.83 ± 1.75[b,c]	9.67 ± 3.62[c]	11.20 ± 2.25[c]	**0.0087***	**0.46 (0.0008)**
Total cholesterol	186.60 ± 4.09 (135–257)	177.50 ± 6.27	181.09 ± 8.88	190.33 ± 7.59	203.30 ± 9.96	0.1335	0.27 (0.0607)
Triglycerides[e]	83.36 ± 5.46 (36–204)	70.94 ± 7.44	90.75 ± 13.39	86.33 ± 12.50	90.80 ± 11.34	0.4795	0.17 (0.2253)
HDL cholesterol	58.02 ± 1.64 (35–83)	58.63 ± 3.53	57.00 ± 3.43	58.00 ± 3.12	58.30 ± 2.82	0.9873	0.003 (0.9824)
LDL cholesterol	111.90 ± 3.69 (54–174)	104.69 ± 5.37	105.92 ± 9.05	115.00 ± 5.33	126.90 ± 9.56	0.1477	**0.28 (0.0464)**
No. risk factors (7 possible)	2.96 ± 0.18 (1–5)	2.25 ± 0.36[b]	2.75 ± 0.33[b,c]	3.58 ± 0.15[c]	3.60 ± 0.34[c]	**0.0072***	**0.45 (0.0011)**
n (% yes)							
Age >45 yr	50 (100%)	16 (100%)	12 (100%)	12 (100%)	10 (100%)	–	
BMI >25 kg·m^{-2}	18 (36%)	4 (25%)	3 (25%)	7 (58%)	4 (40%)	0.2470	
Tobacco current or previous use	24 (48%)	4 (25%)	4 (33%)	6 (50%)	10 (100%)	**0.0016***	
Family history of heart disease	21 (42%)	4 (25%)	7 (58%)	5 (42%)	5 (50%)	0.3239	
History of hypertension	12 (24%)	4 (25%)	4 (33%)	4 (33%)	0 (0%)	0.2296	
History of diabetes	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	–	
History of hyperlipidemia	23 (46%)	4 (25%)	3 (25%)	9 (75%)	7 (70%)	**0.0100***	

Compared with male marathoners with no CAC, male marathoners with moderate and extensive CAC were older ($P = 0.0018$), started running at an older age ($P = 0.0028$), were older when they ran their first marathon ($P = 0.0055$), and had more of the risk factors for CAC examined in this study ($P = 0.0072$). Compared with the male marathoners with no CAC, male marathoners with extensive CAC reported more pack-years of smoking ($P = 0.0087$). In addition, previous tobacco use ($P = 0.0016$) and prevalence of hyperlipidemia ($P = 0.0100$) significantly differed by CAC group, with those with more CAC having higher rates of both. There were no other statistically significant differences between the four groups. Boldface indicates statistically significant findings.

[a]ANOVA overall *F*-test or chi-square test, $^*P < 0.05$.

[b,c,d]Pairwise comparisons are within rows; values with the same superscript do not differ.

[e]Distribution right skewed, using the log-transformed variable, did not change the *t*-test results; untransformed values are reported.

of the year, 20 reported strength training, and 34 had alternate activities including cycling (24), swimming (10), walking/hiking (7), Nordic skiing (5), and snowshoeing (3). Two men ran ultramarathons, one competed as an Ironman™ Triathlete, and one reported cycling 1500 miles a year. The usual weekly training mileage ranged from 15 to 70 (mean = 37.4, median = 33), the peak weekly training miles ranged from 30 to 175 (mean = 65.9, median = 60), and recent training mileage ranged from 0 to 84 (mean = 30.4, median = 27). One subject had stopped running after the 2006 marathon due to hip problems and eventual hip replacement. The reported weekly training mileage for each CAC subcohort was similar with the range (mean/median) of 15–70 (37.2/30) for the no CAC group, 17–60 (32/27) for the low group, 25–55 (40.3/40) for the moderate group, and 20–70 (40.7/40) for the extensive group.

TABLE 2. Differences in age, running characteristics, and health measures by presence or absence of CAC.

(Mean ± SE)	CAC Absent	CAC Present	Difference	P^a
n	16	34		
CAC score	0.00 ± 0.00	402.9 ± 107.26	402.9 ± 157.2	<**0.0001***
Age (yr)	55.38 ± 1.38	61.35 ± 1.09	5.98 ± 1.85	**0.0022***
Age started running (yr)	22.81 ± 1.98	30.56 ± 1.29	7.75 ± 2.32	**0.0016***
Age of first marathon (yr)	28.00 ± 1.55	33.50 ± 1.10	5.50 ± 1.93	**0.0064***
Total marathons completed[b]	78.63 ± 11.46	66.24 ± 6.18	−12.39 ± 11.94	0.3046
BMI (kg·m^{-2})	23.27 ± 0.58	24.58 ± 0.52	1.31 ± 0.86	0.1351
Pack-years of smoking, includes never-smokers	1.94 ± 1.06	7.71 ± 1.65	5.77 ± 2.52	**0.0265***
Total cholesterol (mg·dL^{-1})	177.50 ± 6.27	190.89 ± 5.14	13.39 ± 8.65	0.1283
Triglycerides (mg·dL^{-1})	70.94 ± 7.44	89.21 ± 7.07	18.27 ± 11.52	0.1194
HDL cholesterol (mg·dL^{-1})	58.63 ± 3.53	57.74 ± 1.78	−0.89 ± 3.54	0.8029
LDL cholesterol (mg·dL^{-1})[b]	104.69 ± 5.37	115.29 ± 4.74	10.61 ± 7.84	0.1826
No. risk factors (7 possible)	2.25 ± 0.36	3.29 ± 0.17	1.04 ± 0.35	**0.0045***
n (% yes)				
Age >45 yr	16 (100%)	34 (100%)	–	–
BMI >25 (kg·m^{-2})	4 (25%)	14 (41%)	–	0.2663
Tobacco current or previous use	4 (25%)	20 (59%)	–	**0.0255***
Family history of heart disease	4 (25%)	17 (52%)	–	0.0786
History of hypertension	4 (25%)	8 (24%)	–	0.9096
History of diabetes	0 (0%)	0 (0%)	–	–
History of hyperlipidemia	4 (25%)	19 (56%)	–	**0.0410***

Compared with male marathoners with no CAC, male marathoners with CAC were 5.98 ± 1.85 yr older ($P = 0.0022$); started running at an older age, by 7.75 ± 2.32 yr ($P = 0.0016$); were older when they ran their first marathon, by 5.50 ± 1.93 yr ($P = 0.0064$); reported 5.77 ± 2.52 more pack-years of smoking ($P = 0.0265$); were more likely to have used tobacco (59% vs 25%, $P = 0.0255$); were more likely to have hyperlipidemia (56% vs 25%, $P = 0.0410$); and had 1.04 ± 0.35 more of the risk factors for CAC examined in this study ($P = 0.0045$). There were no other statistically significant differences between the two groups.

[a]Two-sample *t*-test or chi-square test, $^*P < 0.05$.

[b]Distribution right skewed, using the log-transformed variable did not change the *t*-test results; untransformed values are reported.

The CACS ranged from 0 to 3153. The low, moderate, and extensive CAC groups all had significantly greater plaque formation than the group with no CAC. There was only one runner (CAC 3153) with severe coronary artery stenosis documented with a coronary angiogram, and 38 had no stenosis on the CCTA. Two runners in the extensive CAC group had mild-moderate stenosis, five runners in the moderate CAC group had minimal to mild stenosis, and four runners in the low CAC group had minimal to moderate stenosis (two minimal-mild, two moderate).

Table 2 compares the differences in age, running characteristics, and health measures based on the absence or presence of CAC. Again, men with any CAC were older, started running and ran their first marathon at an older age, smoked more tobacco, had more CAD risk factors, and had more hyperlipidemia history than the runners with no plaque. There was a large difference in the rate of family history of CAD in the group with CAC present compared the group with no CAC, although this was not a statistically significant difference (52% vs 25%, $P = 0.08$). In both tables, after adjusting for age, the age of first marathon and the age subjects started running were no longer statistically significantly associated with CAC.

The runner with CACS of 3153 did not complete the CCTA study and underwent a same-day exercise stress echocardiogram because of his high score. This study showed severe ventricular ectopy. A subsequent coronary angiogram showed severe mid right coronary artery stenosis that was treated with coronary artery angioplasty and stent placement. He is included in the risk factor analysis. His story was presented in the local newspaper (3); he had not missed a day of running from age 39 yr until the day of his stent placement at age 65 yr. He reported a 20-yr history of hyperlipidemia, but he had not been on statins. His total cholesterol measured as part of the study was 202 $mg \cdot dL^{-1}$ with an HDL cholesterol of 51 $mg \cdot dL^{-1}$ and an LDL cholesterol of 136 $mg \cdot dL^{-1}$. He started another "running streak" 2 d after his procedure and died 7 yr later at age 72 yr. He is the only known death in this cohort.

DISCUSSION

This cohort of marathon runners was recruited from the Twin Cities Marathon Charter Club (participants who had run every race since the 1982 inaugural event through the 2006 race). When this group of 50 men was compared with a sedentary group of similar age, the mean coronary plaque volume was significantly greater and suggested that coronary artery plaque volume may be associated with high-volume endurance running (12). However, when this cohort is stratified by CACS, there does not appear to be a relationship between CACS and volume of marathon running, but rather the number and the extent of coronary risk factors (including age) seem to play a major role in the development of CAC within this cohort. These results are similar to our women's study (11). Of interest, there was no severe stenosis in any of the male runners except for the individual with a CACS >3000 who started running at age 39 yr to counter his previous cardiac health risk factors.

There were significant differences between age, the age started running, and the age of first marathon between the no CAC group and both the moderate and extensive CAC cohorts. The no CAC cohort was not only younger but started both running and running marathons at younger ages. One runner in the no CAC group began running at age 7 yr. The sum of risk factors, current or previous tobacco use, pack-years of smoking, and personal history of hyperlipidemia were significantly greater in the moderate and extensive CAC groups compared with the runners with no CAC. There were clearly significant differences in age, age starting running, and age of first marathon between the subcohort with no coronary calcium compared with the runners with any CAC and the subcohorts with either moderate or extensive CAC. The younger males in this cohort may have had healthier lifestyles than their counterparts in their 20s and 30s, and as a result benefited with no or lower CACS. In both tables, after adjusting for age, the age of first marathon and the age started running were no longer statistically significantly associated with CAC. This may indicate that these relationships are a function of age at the time of the study rather than the age they started running.

In sedentary men and women, increased calcified and noncalcified coronary artery plaque formation have been associated with increasing age, family history, tobacco use, hypertension, hyperlipidemia, diabetes mellitus, and obesity. These associations likely hold true in runners also. Of the 50 runners, 16 (32%) had no coronary plaque and over 50% had CACS <100. Although 72% of the cohort had an abnormal CAC, most of these men had no evidence of coronary stenosis. It is important to remember that an elevated calcium score reflects the presence of calcium or noncalcified plaque, but the calcium does not necessarily obstruct the artery. However, the presence of plaque does increase the future risk for a myocardial infarction. The no-CACS cohort had the least risk factors. It is likely that the no-CACS cohort had adapted healthier lifestyles earlier in life, contributing to lower CAD finding in our study. Of the 34 runners (68%) with CAC, only 10 were on statin medications before the study, suggesting their health profiles were considered low risk for CAD, possibly due to their high levels of activity. In the any CAC present cohort, many subjects likely had risk factors for CAD before starting running, including previous tobacco use.

Comparing the runners with no CAC to the other subcohorts and to the CAC present cohort, there is a significant association between age, age starting running, age of first marathon, history of tobacco use, history of hyperlipidemia, and sum of cardiac disease risk factors. However, there were no significant differences in the number of marathons completed or average BMI. These associations suggest that past lifestyle choices play a greater role in CACS than the volume of marathon running as the men with the

most plaque started running later in life, possibly to correct diet, exercise, weight, and smoking concerns. Every runner in the extensive CAC group had been a smoker with an average of 12 pack-years. Family history of CAD was fairly evenly distributed among all four groups but was trending toward statistical significance when CAD was present in a runner. Ironically, history of hypertension was present in all the groups except the extensive CAC group.

There was very little difference in HDL cholesterol between the groups, which is surprising as HDL seems to be a prominent risk factor in the general population. On the National Heart, Lung, and Blood Institute, 10-yr coronary event calculator based on the Framingham Study, HDL cholesterol <40 $mg \cdot dL^{-1}$ is considered a major risk factor for heart disease and HDL cholesterol >59 $mg \cdot dL^{-1}$ is considered protective against heart disease (7). The average HDL cholesterol for the group as a whole was 58 ± 1.6 $mg \cdot dL^{-1}$, and the regular high-volume running may be of benefit for HDL level. The total cholesterol was significantly greater in the extensive lesion group, and the LDL cholesterol was nearing significance compared with the no CAC group. There was no significant difference in the number of years running, so starting earlier in life may be protective or may imply a healthier lifestyle at an earlier age.

The men in this study had higher CACS than the women marathons we studied (11). Men are also at higher risk than women for sudden cardiac arrest and sudden cardiac death (SCD) during and immediately after a marathon race (4,5,9,10). The relative risk of SCD for men compared with women is in the range of 5–6 (4,10). CAD is the main cause for marathon-related sudden cardiac arrest/SCD in men older than 40 yr (4,10). Some of this difference may be due to lifestyle factors at a younger age manifesting as unstable plaque in the +40-yr age ranges. Lifestyle including diet and physical activity decisions made in the young adult years may affect cardiac risk despite a high-volume running program later in life.

There are several limitations to this study. This was a convenience sample of men who volunteered for the protocol. Both the cohort and the subcohorts are small sample sizes. The survey required self-report, and recall may not have been complete or entirely accurate. There is no control group with CACS and survey results. The risk factor analysis combined current and past smokers and pooled overweight with the obese men based on BMI. BMI may not be the best marker for overweight status in physically active men as a higher percentage of more dense lean muscle tissue may drive the BMI calculation more than 25 $kg \cdot m^{-2}$ and inappropriately mislabel some of the runners as overweight. There may also be selection bias as those runners who developed advanced CAD and did not make the 25-yr mark with the Charter Club are not included in the study. In addition, neither the number of marathons completed nor the year they started running marathons reflects the actual amount or intensity of training for either the individuals or the cohort.

CONCLUSION

Among experienced male marathoners who have run marathons for 26–34 yr and completed between 27 and 171 marathons each, CACS and CAD are related to CAD risk factors (i.e., age and smoking) and not the number of marathons run or years of running. This suggests that among lifelong endurance athletes, more endurance exercise is not associated with an increased risk of coronary artery plaque formation.

This work was supported in part by the Ken Rome Foundation, Minneapolis, MN http://kenrome5k.wordpress.com/2008/08/01/ken-rome-foundation-information. The authors have no conflicts of interest to disclose. The results of the present study do not constitute endorsement by the American College of Sports Medicine. The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

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